POSSIBLE MECHANISIM FOR MODULATING CARDIOVASCULAR SYSTEM DURING RUNNING IN HUMANS

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Abstract-**Cardiovascular response to running exercise was studied with a simple pulsatile cardiovascular model. Published experimental data during graded exercise were used to tune the model parameters. Study results show that cardiac output is modulated by rhythmic muscle contractions during running. It oscillates when step rate and heart rate are not synchronised but stabilises when they are synchronised. A maximum cardiac output can be achieved by synchronising step rate with heart rate at an optimal phase delay.**

Keywords – **Cardiovascular model, heart rate, step rate, running**

I. INTRODUCTION

Long distance running requires a high level cardiovascular performance to supply oxygenated blood to metabolizing muscles over long periods of time. A large cardiac output is the major factor distinguishing champion endurance athletes from other well-trained athletes. Researches from [3][4][5] suggest that synchronization between the heart rate and the step rate during running may favor an athlete with moderate increase in cardiac output. To further explore it, we did a simulation study in order to get some insight into this phenomenon. In this paper, a simple pulsatile cardiovascular model at rest is presented firstly. Then the parameters of the model are tuned so that the output from the model matches published experimental data under exercise condition. Finally the model is used to simulate the cardiovascular responses to running exercise with the step rate synchronized with the heart rate.

II. THE DESCRIPTION OF THE MODEL

The basic model is a modified version based on [1]. It is simplified to contain only the minimum five compartments required for a pulsatile model (see Fig. 1). Parameters are distinguished by the indices 'sa', 'sp', 'sv', 'la' and 'lv' to represent systemic arteries, systemic peripheral regions, systemic veins, left atrium and left ventricle respectively. The vascular compartments include systemic arteries (sa), systemic peripheral regions (sp) and systemic veins (sv). The systemic peripheral regions represent the arterioles, the capillary region and the venules in the systemic part of the circulatory system. The heart contains the left atrium (la) and left ventricle (lv). Each compartment is modeled by two components (Fig. 2): a resistance R and a capacitance C. R represents the pressure loss and energy dissipation along the vessels in the vascular compartments and the valves in the heart. C denotes the amount of blood the compartment can hold under a particular pressure. P represents the blood pressure and F is the blood flow.

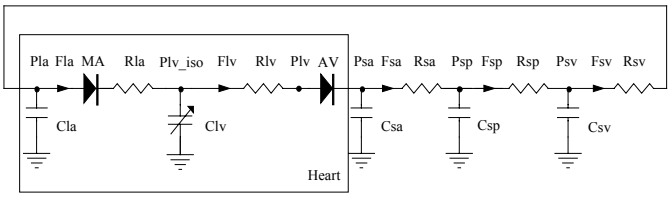

Fig. 1. The electrical analogue model of cardiovascular system.

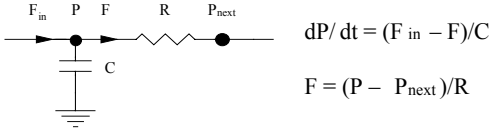

Fig. 2. Dynamic function for one compartment.

The isometric left ventricular pressure Plv_iso at any given time is approximated by both end-diastolic pressure-volume relationship (EDPVR) and end-systolic pressure-volume relationship (ESPVR) as follows:

$$P_{lv\_iso} = \alpha P_{ESPVR} + (1-\alpha) P_{EDPVR} \quad (1)$$

$$P_{EDPVR} = B(e^{AV_{lv}} - 1) \quad (2)$$

$$P_{ESPVR} = E_{\max}(V_{lv} - V_0) \quad (3)$$

E_{max} is the slope of the ESPVR. α is an activation function which starts from 0 at end-diastolic point, and reaches its maximum value 1 at end-systolic point and returns to zero at the end of the systole period.

$$\alpha = \begin{cases} \sin^2(\frac{T}{T_s}\mu\pi), & 0 \le \mu \le \frac{T_s}{T} \\ 0, & \frac{T_s}{T} \le \mu \le 1 \end{cases} \quad (4)$$

μ is a dimensionless variable, increments from 0 to 1 in each cardiac cycle.

$$\mu = \frac{t}{T} \mod 1 \quad (5)$$

$$T = \frac{60}{HR} \quad (6)$$

HR is heart rate in beat/min. The duration of systole Ts changes linearly with heart rate.

$$T_s = T_{s0} - \frac{k_s}{T}. \quad (7)$$

Since the system is closed and blood flow only occurs between compartments, the total blood volume in the cardiovascular system (CVS) is constant.

$$V_t = C_{la}P_{la} + V_{lv} + C_{sa}P_{sa} + C_{sp}P_{sp} + V_{sv} + V_u \quad (10)$$

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Abstract

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Vu represents the total unstressed volume. Therefore, the venous volume can be obtained directly as follows:

$$V_{sv} = V_t - C_{la}P_{la} - V_{lv} - C_{sa}P_{sa} - C_{sp}P_{sp} - V_u \qquad (11)$$

III. PARAMETER TUNING UNDER EXERCISE

In order to simulate the cardiovascular system during exercise, the published experimental data (see Table 1) by Plotnick et al [2] is used to tune the parameters of the model. HR is the heart rate. Psys is the arterial systolic pressure. Vsys and Vdia are the systolic and diastolic left ventricular volume. The main idea is to have the model reproduce these expected independent results by adjusting the parameters of the model. The input to the model is the heart rate HR corresponding to each workload. The outputs of the model will be just Psys, Vsys and Vdia since other values are derived from them. Since Emax and the unstressed volume Vu are under vigal control and Rsp is auto-regulated to guarantee a sufficient blood flow in the relevant muscles under exercise, these three parameters are chosen to be the tuning parameters. For each workload, the heart rate input is known, the model output values are compared with the expected experimental data in Table 1. Parameter Emax, Rsp and Vu are tuned to bring the difference to a minimum. The rule for tuning these parameters is obtained by analysing the effects of each parameter. Since Emax is the ratio of the left ventricular end-systolic pressure to the end-systolic volume Vsys, increasing Emax will increase the left ventricular end-systolic pressure or/and decrease Vsys. And increasing the left ventricular end-systolic pressure will also raise Psys. The first tuning rule can be obtained from this relationship: reducing Emax if Psys is higher and Vsys is lower than their expected values. Besides, it can be seen from Figure 1 that reducing the peripheral resistance Rsp will reduce the difference between Psp and Psv. Consequently it will cause Psp to drop and Psv to rise. The drop in Psp will in turn reduce the arterial systolic pressure Psys and the rise in Psv will also raise the left ventricular diastolic volume Vdia. Therefore our second tuning rule should be that reducing Rsp if Psys is higher and Vdia is lower than the expected values. Finally, since the unstressed volume Vu is the blood volume not circulating in the CVS, reducing Vu actually allows more blood into the circulation loop. This will effectively raise both pressure and volume of each compartment in CVS. Therefore our last tuning rule is that reducing Vu if Psys, Vsys and Psys are all higher than their expected values. All three rules for tuning the parameters are shown in Fig. 3. These tuning rules have included all possible conditions of the three output values. The magnitude of the adjustment can be large if it doesn't change the current condition and should be very small if it changes the current condition.

The tuned parameters at each workload are shown in Fig. 4. It can be seen that the resistance and unstressed volume are roughly constant once the exercise has started. Only Emax increases dramatically at high workload. Therefore the following

TABLE I
EXPERIMENTAL DATA DURING PROGRESSIVE EXERCISE ADAPED FROM[2]

Workload (W)	rest	25	50	75	100	125-250
HR (bpm)	73	97±2	104±2	116±2	128±3	166±3
Psys (mmHg)	126±2	147±2	154±2	167±3	183±4	204±5
Vdia (ml)	132±6	155±9	157±9	155±9	147±9	137±9
Vsys (ml)	48±3	46±4	43±4	37±4	31±3	24±3

IF			THEN		
Psys	Vsys	Vdia	Emax	Rsp	Vu
↑	↓		↓		
↓	↑		↑		
↑		↓		↓	
↓		↑		↑	
↑	↑	↑			↑
↓	↓	↓			↓

↑ : increase; ↓: decrease; space: ignor

Fig. 3. Tuning rules for estimating model parameters.

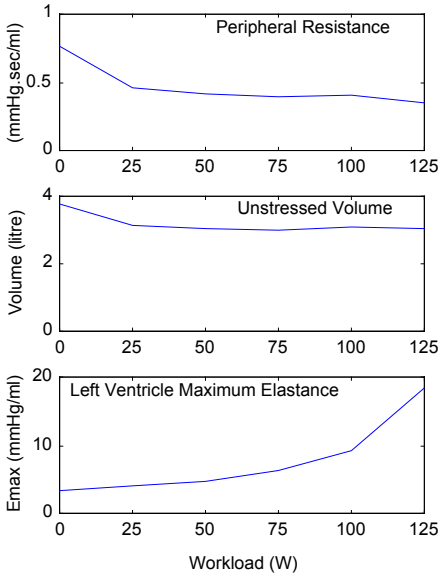

Fig. 4. Model parameters at each workload

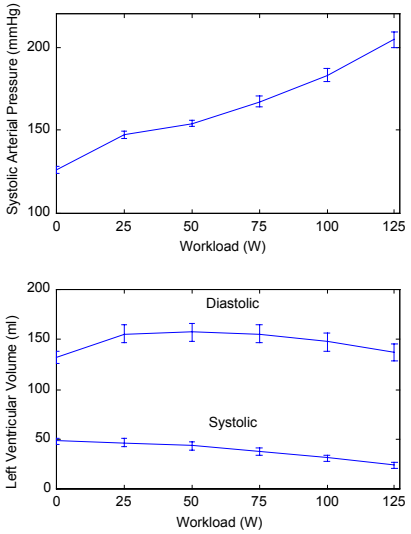

Fig. 5. Simulation results (line) compared with experimental data (bar)

simulation study was conducted at 100W workload. Fig. 5 shows the comparison between the simulation outputs and the experimental data.

IV. SIMULATION

Rhythmic muscle contraction in legs temporarily interrupts the blood supply to the muscle tissue thereby impeding the blood flow in the capillaries, but enhances the venous return to the heart due to the muscle pump effect [6][7].

The muscle pump effect, which refers to the mechanism of repeatedly emptying the veins towards the heart by rhythmic muscular contraction, is of great circulatory importance in the legs. It promotes the venous return to the heart and prevents the pooling of large amounts of blood in the venous system. At rest, the venous system contains 65 to 70 percent of the total blood volume. By constriction of the venules and veins, close to half of that blood volume may be mobilized and emptied toward the heart [7]. It has been calculated that muscle pumping of blood by the lower limbs contributes more than 30% of the energy required to circulate blood during running [6]. Moreover, intramuscular pressure rises during exercise. Complete occlusion of the blood flow occurs at 20-30% of the maximum voluntary contraction in the calf muscles and up to 1,025 mmHg intramuscular pressure had been measured during maximum isometric muscle contraction in human [5]. The blood flow to the muscle is reduced when the contracting muscle compresses the vessels. But in periods between contractions, an increased perfusion pressure would result in an important gain in blood supply to the muscle.

We simulated this muscle pumping and squeezing effects by modulating the unstressed volume in the veins. Reducing the unstressed volume increases the venous pressure. This increased venous pressure will fill the heart with more blood, which is equivalent to the contraction phase of the muscle pump. The increased venous pressure also hinders the blood flow in the peripheral region. This is equivalent to blocking the blood flow in the muscle tissues when squeezing the muscle. The modulating signal is defined as:

$$V\,\mathrm{mod} = \begin{cases} Vm\sin(2\pi\lambda+\varphi), & 0 \le \lambda \le \frac{1}{2} \\ 0, & \frac{1}{2} \le \lambda \le 1 \end{cases} \quad (12)$$

λ is a dimensionless variable, increments from 0 to 1 in each step cycle. φ is the phase delay.

$$\lambda = \frac{t}{T_{SR}} \mod 1 \quad (13)$$

$$T_{SR} = \frac{60}{SR} \quad (14)$$

SR is the step rate in step/min. Fig. 6 shows the waveform of the modulating signal Vmod with amplitude Vm = 1 and phase delay φ = 0. The foot touches ground at t = 0 (and T_{SR}) and lift off ground at t = $0.5T_{SR}$ (and $1.5T_{SR}$).

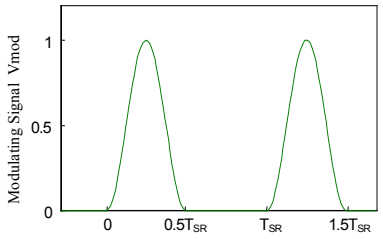

Fig.6. Modulating signal waveform.

In order to investigate only the effects of the rhythm of the step rate on the cardiovascular performance, the mean of the modulating signal within one cycle should be zero. To achieve this, the integral of the modulating signal in one cycle is subtracted from the modulating signal. Therefore (11) becomes

$$V_{sv} = V_t - C_{la}P_{la} - V_{lv} - C_{sa}P_{sa} - C_{sp}P_{sp} - (V_u - V_{um}) \quad (11)$$

$$V_{um} = V_{\mathrm{mod}} - \int_0^1 V_{\mathrm{mod}}\,d\lambda \quad (16)$$

We chose Vm = 530ml which is 10% of the total blood volume. Since we only need qualitative results, the absolute value is not very important. Fig. 7 shows the simulated stroke volume when the step rate is different from and equal to the heart rate. The closer the step rate approaches the heart rate, the slower the stroke volume oscillates. Once the step rate equals the heart rate, the stroke volume is constant and is decided by the phase difference between the step rate and heart rate signals (see Fig. 8). It can be seen that once the step rate equals the heart rate, a constant maximum stroke volume can be achieved if we can keep the phase to an optimal point. If the cardiac output is maintained as constant, the heart rate would be higher or lower at different phase delays in order to obtain the same cardiac output. (see Fig. 9).

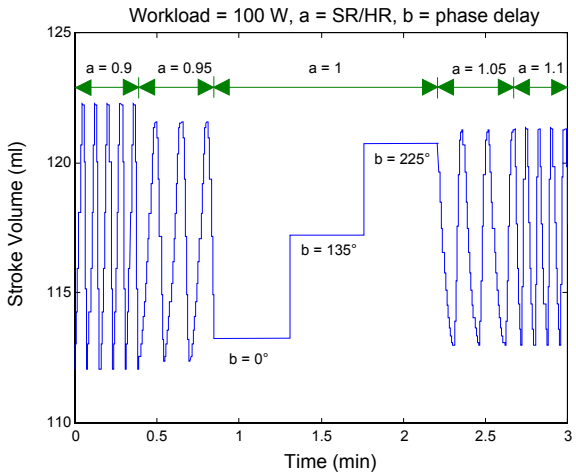

Fig. 7. Stroke volume is modulated by the difference between step rate and heart rate. When the step rate is synchronized with the heart rate, the stroke volume is constant and the value is phase dependent. a = step rate/ heart rate. b = phase delay when the step rate is synchronized with the heart rate.

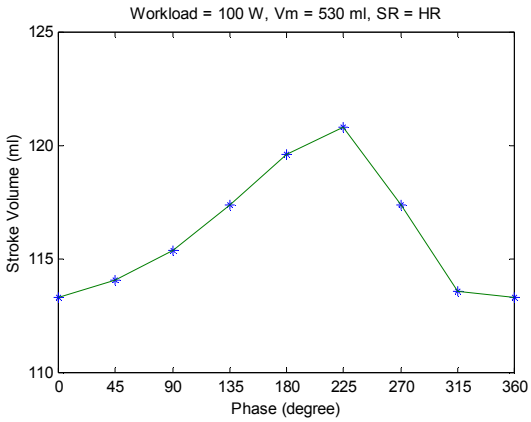

Fig. 8. Stroke volume against phase delay while step rate is synchronized with heart rate.

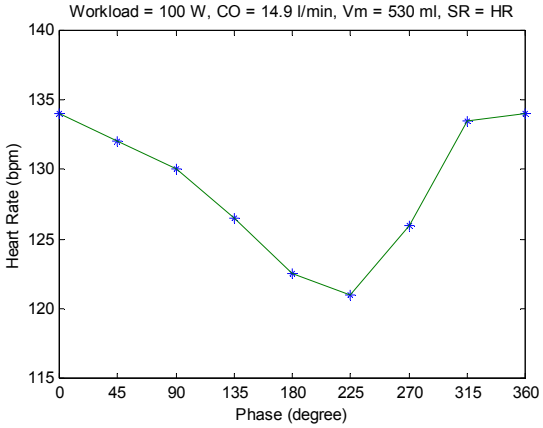

Fig. 9. Heart rate against phase delay while step rate is synchronized with heart rate and the cardiac output is kept constant.

IV. DISCUSSION AND CONCLUSION

Results from this simulation study show that the overall cardiac performance depends on whether step rate is synchronized with heart rate or not. When step rate is not synchronized with heart rate, the stroke volume is modulated by the difference between the step rate and the heart rate. Once the step rate is synchronized with the heart rate, the stroke volume is determined by how much the step lags the heartbeat. If the step rate is synchronized with the heart rate and the phase delay is kept constant, the stroke volume is also constant. It can be seen from Fig. 8 that the maximum stroke volume is reached when the phase delay is 225 degrees and minimum at 0 degree. It is not clear how much unstressed blood volume can actually be modulated during running. Since the venous system contains 65 to 70 percent of the total blood volume at rest and close to half of the venous blood volume may be mobilized by muscle constriction of the venules and veins during running [7], it is likely that the amplitude of modulation in the unstressed volume may be more than just 10% of the total blood volume. For the same cardiac output, if step rate is synchronized with heart rate, the heart rate will vary depending on the phase delay (Fig. 9). By synchronizing step rate with heart rate at an optimum phase delay, a constant stroke volume can be achieved at its maximum capacity. It seems possible that a long distance runner may further extend his or her maximum cardiac output by using this technique.

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